

Naval Health Research Center Detachment (Toxicology)

Feb 2003

DISTRIBUTION STATEMENT A

Approved for Public Release
Distribution Unlimited

**Risk Report on Perfluorooctanesulfonate (PFOS) as a Component of Mist
Suppressants in Chrome-Plating Tanks**

TOXDET-03-05

Andrew J. Bobb¹, Ph.D., USNR
Kenneth R. Still, Ph.D., msc, USN

1. Naval Health Research Center Detachment (Toxicology) – NHRC/TD

Bldg 433
2612 5th St.
Wright-Patterson AFB, OH 45433-7903

Correspondence to LT Andrew J. Bobb at NHRC/TD



20030306 072

Risk Report on Perfluorooctanesulfonate (PFOS) as a Component of Mist Suppressants in Chrome-Plating Tanks

Andrew J. Bobb, PhD, LT MSC USNR
Kenneth R. Still, Ph.D, MSC, USN



Naval Health Research Center Detachment, Toxicology
Wright-Patterson AFB, OH

February 2003

(This page intentionally left blank)

ABSTRACT

Perfluorooctanesulfonate (PFOS) is a synthetic perfluorinated surfactant recently discovered to be ubiquitous in the environment. Animal data suggest a high tolerance for PFOS, as does epidemiological analysis of workers in PFOS manufacturing plants. A suggested reference dose of 0.02 mg/kg/day is presented. This constitutes an unlikely exposure level in the application of interest, as a component of mist suppressant in chromium plating tanks.

(This page intentionally left blank)

PFOS: A UBIQUITOUS FLUOROCARBON

Perfluorooctanesulfonate (PFOS) and Perfluorooctanoic acid (PFOA) are structurally and chemically related synthetic perfluorinated surfactants used in a number of industrial applications, including plasticizers, lubricants, wetting agents, etc. The current application of interest to the Navy is the use of PFOS in mist suppressants, preventing the release of aerosols from chromium plating tanks. PFOS/PFOA also appear to be the metabolic product of breakdown of other xenobiotic compounds (Olsen et al, 1999). Recent reports have suggested that PFOS is nearly ubiquitous in the environment (Giesy and Kannan, 2001; Kannan et al 2001a; Kannan et al 2001b), and that it may bioaccumulate at higher levels in the food chain (Giesy and Kannan, 2001). Commercially available human serum (presumably without occupational exposure to PFOS) contains an average PFOS concentration of 24 ppb (Hansen et al, 2001)

RODENT DATA

Toxicity data from rodents suggests a high potential for liver toxicity for both compounds, and some evidence for developmental toxicity. Inhalation of the ammonium salt of PFOA at 8 or 84 mg/m³ results in liver-weight increases and microscopic liver necrosis in rats (Kennedy et al, 1986). No published data on inhalational PFOS exposure is available. Rats gavaged with up to 50 mg/kg/day ammonium PFOA had significant increases in estrogen, and decreases in testosterone (Cook et al, 1992). Rats which were fed PFOA or PFOS exhibited reduced cholesterol synthesis and reduced serum triacylglycerides (Haughom and Spydevold, 1992). *In utero* exposure to PFOS at levels up to 1.0 mg/kg/day had no effect on rabbit pups up to the time of birth (Case et al, 2001a); but rat pups born to dams fed 1.6 mg/kg/day exhibited high infant mortality (Case et al 2001b).

HUMAN DATA

Humans have been regularly exposed to PFOA and PFOS in industrial synthesis plants. An epidemiological study of 2788 male and 749 female workers employed in a PFOA synthesis plant between 1947 and 1983 (Gilliland and Mandel, 1993) exhibited no significant deviations from unexposed individuals, except for a possible increase in prostate cancer deaths (4 deaths in exposed workers, 2 in unexposed). Another study of 115 occupationally exposed workers found no changes in hepatic enzymes, lipoproteins and cholesterol (Gilliland and Mandel, 1996). Another study of a total of 191 occupationally-exposed workers (performed in two different years) found no significant effect of PFOA on human hormone levels (Olsen et al, 1998); a similar study with PFOS using 317 male workers found no effects on serum hepatic enzymes, cholesterol, or lipoproteins (Olsen et al 1999). The half-life of PFOA in human systems is estimated to be 18 to 24 months (Ubel et al, 1980) and the half-life of PFOS may be even longer (Olsen et al, 1999).

APPLICATION AND CONCLUSIONS

There exists significant contradiction between the rodent and human data for PFOS/PFOA exposure. A potent liver toxicant in rodents should produce some level of toxicity in humans, particularly over the long exposure times; therefore it may be that the toxicity seen in rodents is the result of a mechanism which is not active in humans. This is not unprecedented; saccharin causes bladder tumors in rats (Reuber 1978) yet epidemiological data demonstrate that it is clearly noncarcinogenic in humans (Elcock and Morgan, 1993). Specific mechanisms exist in some animals, particularly in response to high-dose exposure, that render extrapolation between species impossible, for a particular effect (Cohen, 1995; Whysner and Williams, 1996).

PFOS is a component of mist suppressants used in chrome plating tanks. The primary hazard in such applications is hexavalent chromium a known carcinogen. Analysis of plating tank contents (Naval Facilities Engineering, Personal Communication; testing done by Centre Analytical Laboratories, Inc., State College, PA) indicates a concentration of <37 mg/L. PFOS has a very low volatility (so much so that it has not been possible to obtain vapor inhalational toxicology data), therefore it is likely that the only airborne exposure will come from process-generated aerosols.

Given a lack of human exposure data (apart from cumulative serum levels) it is impossible to compare the animal and human data, or to derive a safe exposure level solely from the industrial exposure data. Both the liver toxicity and the potential reproductive toxicity (changes in hormone levels) exhibited in animal exposure data are specifically contradicted by human epidemiological data. There is, however, no evidence to suggest that the animal developmental toxicity data is inapplicable to humans. It seems therefore most conservative to base toxicity profiles on this data. The NOAEL is 1.0 mg/kg/day in rabbits (Case et al, 2001a). Multiplying by an interspecific uncertainty factor of 10 and an intraspecific uncertainty factor of 5 (reduced from 10 because the epidemiological data suggest similar response to this compound between males and females- Gilliland and Mandel, 1993), we would derive a maximum daily dose of 0.02 mg/kg/day. For a 70 kg individual, therefore, the recommended limit would equate to drinking ~35 mL of tank contents, an unlikely exposure level. Furthermore, personnel likely to be exposed to PFOS from tanks or process-generated aerosols will be co-exposed to hexavalent chromium at much higher concentrations, and with much more serious health consequences. Measures in place to monitor or control chromium exposure will be more than adequate to protect the health of workers from PFOS, and that PFOS in chrome plating tanks will not significantly increase the risk of heath consequences, barring any unforeseen complications of co-exposure.

LITERATURE CITED

Case MT, York RG, Christian MS. 2001a. Rat and rabbit oral developmental toxicology studies with two perfluorinated compounds. *Int J Toxicol* 20:101-109

Case MT, York RG, Buttenhoff JL. 2001b. Oral (gavage) cross-fostering study of potassium perfluorooctanesulfonate (PFOS) in rats. *Toxicologist* 60:221-222

Cohen SM. 1995. Human relevance of animal carcinogenicity studies. *Regul Toxicol Pharmacol* 21: 75-80

Cook JC, Murray SM, Frame SR, Hurt ME. 1992. Induction of Leydig cell adenomas by ammonium perfluorooctanoate: a possible endocrine-related mechanism.

Elcock M, Morgan RW. 1993. Update on artificial sweeteners and bladder cancer. *Regul Toxicol Pharmacol* 17: 35-43

Giesy JP, Kannan K. 2001. Global distribution of perfluorooctane sulfonate in wildlife. *Environ Sci Technol* 35: 1339-1342

Gilliland FD, Mandel JS. 1993. Mortality among employees of a perfluorooctanoic acid production plant. *J Occup Med* 35: 950-954

Gilliland FD, Mandel JS. 1996. Serum perfluorooctanoic acid and hepatic enzymes, lipoproteins, and cholesterol: a study of occupationally exposed men. *Am J Ind Med* 29: 560-568

Hansen KJ, Clemen LA, Ellefson ME, Johnson HO. 2001. Compound-specific, quantitative characterization of organic fluorochemicals in biological matrices. *Environ Sci Technol*, 35 (4), 766 -770

Haugom B, Spydevold O. 1992. The mechanism underlying the hypolipemic effect of perfluorooctanoic acid (PFOA), perfluorooctane sulphonic acid (PFOSA) and clofibrate acid. *Biochim Biophys Acta* 1128: 65-72

Kannan K, Koistinen J, Beckmen K, Evans T, Gorzelany JF, Hansen KJ, Jones PD, Helle E, Nyman M, Giesy JP. 2001. Accumulation of perfluorooctane sulfonate in marine mammals. *Environ Sci Technol* 35: 1593-1598

Kannan K, Franson JC, Bowerman WW, Hansen KJ, Jones PD, Giesy JP. Pefluorooctane sulfonate in fish-eating water birds including bald eagles and albatrosses. *Environ Sci Technol* 35: 3065-3070

Kennedy GL Jr, Hall GT, Brittelli MR, Barnes, JR, Chem HC. 1986. Inhalation toxicity of ammonium perfluorooctanoate. *Food Chem Toxicol* 24: 1325-1329

Olsen GW, Gilliland FD, Burlew MM, Burris JM, Mandel JS, Mandel JH. 1998. An epidemiological investigation of reproductive hormones in men with occupational exposure to perfluorooctanoic acid. *J Occ Environ Med* 40: 614-622

Olsen GW, Burris JM, Mandel JH, Zobel LR. 1999. Serum perfluorooctane sulfonate and hepatic and lipid clinical chemistry tests in fluorochemical production employees. *J Occ Environ Health* 41:799-806

Reuber MD. 1978. Carcinogenicity of saccharin. *Environ Health Perspect* 25:173-200

Ubel FA, Sorenson SD, Roach DE. 1980. Health status of plant workers exposed to fluorochemicals, a preliminary report. *Am Ind Hyg Assoc J* 41: 584-589

Williams GM, Whysner J. 1996. Epigenetic carcinogens: evaluation and risk assessment. *Exp Toxicol Pathol* 48: 189-95

REPORT DOCUMENTATION PAGE			Form Approved OMB No. 0704-0188
<p>Public reporting burden for this collection of information is estimated to average 1 hour per response, including the time for reviewing instructions, searching existing data sources, gathering and maintaining the data needed, and completing and reviewing the collection of information. Send comments regarding this burden estimate or any other aspect of this collection of information, including suggestions for reducing this burden, to Washington Headquarters Services, Directorate for Information Operations and Reports, 1215 Jefferson Davis Highway, Suite 1204, Arlington, VA 22202-4302, and to the Office of Management and Budget, Paperwork Reduction Project (0704-0188) Washington, DC 20503.</p>			
1. AGENCY USE ONLY (Leave blank)	2. REPORT DATE	3. REPORT TYPE AND DATES COVERED	
	January 2003	N/A	"
4. TITLE AND SUBTITLE Risk Report on Perfluorooctanesulfonate (PFOS) as a Component of Mist Suppressants in Chrome-Plating Tanks			5. FUNDING NUMBERS
6. AUTHOR(S) Andrew J. Bobb, Ph.D. <i>Kenneth Q. Still, Ph.D., MSC USN</i>			
7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES) Naval Health Research Center Detachment Toxicology NHRC/TD 2612 Fifth Street, Building 433 Area B Wright-Patterson AFB, OH 45433-7903			8. PERFORMING ORGANIZATION REPORT NUMBER TOXDET-03-05
9. SPONSORING/MONITORING AGENCY NAME(S) AND ADDRESS(ES) Naval Health Research Center Detachment Toxicology NHRC/TD 2612 Fifth Street, Building 433 Area B Wright-Patterson AFB, OH 45433-7903			10. SPONSORING/MONITORING AGENCY REPORT NUMBER
11. SUPPLEMENTARY NOTES			
12a. DISTRIBUTION AVAILABILITY STATEMENT Approved for public release; distribution is unlimited.		12b. DISTRIBUTION CODE	
13. ABSTRACT (Maximum 200 words) Perfluorooctanesulfonate (PFOS) is a synthetic perfluorinated surfactant recently discovered to be ubiquitous in the environment. Animal data suggest a high tolerance for PFOS, as does epidemiological analysis of workers in PFOS manufacturing plants. A suggested reference dose of 0.02 mg/kg/day is presented. This constitutes an unlikely exposure level in the application of interest, as a component of mist suppressant in chromium plating tanks.			
14. SUBJECT TERMS Perfluorooctanesulfonate, PFOS, mist suppressant			15. NUMBER OF PAGES 9
			16. PRICE CODE
17. SECURITY CLASSIFICATION OF REPORT UNCLASSIFIED	18. SECURITY CLASSIFICATION OF THIS PAGE UNCLASSIFIED	19. SECURITY CLASSIFICATION OF ABSTRACT UNCLASSIFIED	20. LIMITATION OF ABSTRACT UL

GENERAL INSTRUCTIONS FOR COMPLETING SIF 298

The Report Documentation Page (RDP) is used in announcing and cataloging reports. It is important that this information be consistent with the rest of the report, particularly the cover and title page. Instructions for filling in each block of the form follow. It is important to *stay within the lines* to meet *optical scanning requirements*.

Block 1. Agency Use Only (Leave blank).

Block 2. Report Date. Full publication date including day, month, and year, if available (e.g. 1 Jan 88). Must cite at least the year.

Block 3. Type of Report and Dates Covered. State whether report is interim, final, etc. If applicable, enter inclusive report dates (e.g. 10 Jun 87 - 30 Jun 88).

Block 4. Title and Subtitle. A title is taken from the part of the report that provides the most meaningful and complete information. When a report is prepared in more than one volume, repeat the primary title, add volume number, and include subtitle for the specific volume. On classified documents enter the title classification in parentheses.

Block 5. Funding Numbers. To include contract and grant numbers; may include program element number(s), project number(s), task number(s), and work unit number(s). Use the following labels:

C - Contract	PR - Project
G - Grant	TA - Task
PE - Program Element	WU - Work Unit
	Accession No.

Block 6. Author(s). Name(s) of person(s) responsible for writing the report, performing the research, or credited with the content of the report. If editor or compiler, this should follow the name(s).

Block 7. Performing Organization Name(s) and Address(es). Self-explanatory.

Block 8. Performing Organization Report Number. Enter the unique alphanumeric report number(s) assigned by the organization performing the report.

Block 9. Sponsoring/Monitoring Agency Name(s) and Address(es). Self-explanatory.

Block 10. Sponsoring/Monitoring Agency Report Number (If known)

Block 11. Supplementary Notes. Enter information not included elsewhere such as: Prepared in cooperation with ; Trans. of ; To be published in.... When a report is revised, include a statement whether the new report supersedes or supplements the older report.

Block 12a. Distribution/Availability Statement. Denotes public availability or limitations. Cite any availability to the public. Enter additional limitations or special markings in all capitals (e.g. NOFORN, REL, JTAR).

DOD - See DoDD 5230.24, "Distribution Statements on Technical Documents."

DOE - See authorities.

NASA - See Handbook NH13 2200.2.

NTIS - Leave blank.

Block 12b. Distribution Code.

DOD - Leave blank.

DOE - Enter DOE distribution categories from the Standard Distribution for Unclassified Scientific and Technical Reports.

NASA - Leave blank.

NTIS - Leave blank.

Block 13. Abstract. Include a brief (*Maximum 200 words*) factual summary of the most significant information contained in the report.

Block 14. Subject Terms. Keywords or phrases identifying major subjects in the report.

Block 15. Number of Pages. Enter the total number of pages.

Block 16. Price Code. Enter appropriate price code (*NTIS only*).

Blocks 17. - 19. Security Classifications. Self-explanatory. Enter U.S. Security Classification in accordance with U.S. Security Regulations (i.e., UNCLASSIFIED). If form contains classified information, stamp classification on the top and bottom of the page.

Block 20. Limitation of Abstract. This block must be completed to assign a limitation to the abstract. Enter either UL (unlimited) or SAR (same as report). An entry in this block is necessary if the abstract is to be limited. If blank, the abstract is assumed to be unlimited.